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### ★ Polycystic ovary syndrome in adolescents ★

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Polycystic ovary syndrome (PCOS) is the most common cause of infertility in women, frequently becomes manifest during adolescence, and is primarily characterized by ovulatory dysfunction and hyperandrogenism. The syndrome is heterogeneous clinically and biochemically. The diagnosis of PCOS has lifelong implications with increased risk for metabolic syndrome, type 2 diabetes mellitus, and possibly cardiovascular disease and endometrial carcinoma. PCOS should be considered in any adolescent girl with a chief complaint of hirsutism, treatment-resistant acne, menstrual irregularity, or obesity. There are several proposed diagnostic criteria for polycystic ovary syndrome (PCOS). Few studies focus on treatment of PCOS in adolescents, so management is based primarily on studies in adults. Hormonal treatment with estrogen-progestin contraceptives is ordinarily the first-line approach for management of PCOS, in combination with weight management for patients with obesity. Diet and exercise are first-line treatments for obese adolescents with PCOS.

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Polycystic ovary syndrome is a heterogeneous syndrome of unexplained chronic hyperandrogenism and oligo-anovulation and the most common cause of chronic anovulation associated with hyperandrogenic state. The

incidence of it is about 5-10% in reproductive-age women. Diagnosis is made by excluding other hyperandrogenic disorders (e.g., nonclassic and classic adrenal hyperplasia, androgen secreting tumors, hyperprolactinemia) in women with chronic anovulation and androgen excess. Major morbidities are related to reproductive and cardiovascular systems. The most important reproductive morbidities consisting infertility, irregular uterine bleeding, increased pregnancy loss and higher risk of endometrial cancer than non PCO people. Cardiovascular risks are linked to insulin resistance and common occurrence of obesity, although it also occurs in non-obese women with PCOS. PCOS is considered to be a heterogeneous disorder with multifactorial causes and it appears to account for 70% of the variance in pathogenesis. Both heritable and non-heritable factors contribute to arise it. So a positive family history of chronic anovulation and androgen excess increases the risk for occurring PCOs. There are many evidences that risk factors for PCOS can be recognized in childhood. Congenital virilizing disorders; above average or low birth weight for gestational age; premature adrenarche, particularly exaggerated adrenarche; atypical sexual precocity; or intractable obesity with acanthosis nigricans, metabolic syndrome, and pseudo-Cushing syndrome or pseudo-acromegaly in early childhood have been identified as independent prepubertal risk factors for the development of PCOS. During adolescence, PCOS may masquerade as physiological adolescent anovulation. Asymptomatic adolescents with a polycystic ovary occasionally (8%) have subclinical PCOS but often (42%) have a subclinical PCOS type of ovarian dysfunction, the prognosis for which is unclear. Identifying children at risk for PCOS offers the prospect of eventually preventing some of long-term complications associated with this syndrome once our understanding of the basis of the disorder improves.

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